# LOCOMOTOR ATAXIA. TWO CASES: ONE A CASE OF SO-CALLED SPINAL ARTHROPATHY; THE OTHER, ACUTE TABES DORSALIS.[1]

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THE histories of the two cases were as follows:

CASE I.—Mrs. M. R., æt. 40, Germany, housewife, was first admitted to the Presbyterian Hospital September 11th, 1884. Family history negative. Patient commenced to menstruate at 16, and has been regular up to the last few years. At 17 years of age, she married a man who, she stated, "was after fast women." Two years after her marriage, a "fever sore" developed on her lip, which did not heal readily, but did after local and internal medication. At about the same time she had a sore near the anus. This was followed by a sore throat, pain in the bones, and a falling out of the hair, but without an eruption. Patient never had a miscarriage nor a child. Four years after her marriage, patient had a cold and, with this blindness of the right eye, with ptosis.

Since 30, she has been a widow. Her memory appeared to be defective, and it was almost impossible to obtain a satisfactory history.

From the time her husband died until the early part of 1882, she was perfectly well; at that time she commenced to have recurring attacks of dizziness without loss of consciousness, and would fall backwards if unable to catch hold of something for support. The next symptom noticed

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was a feeling as if sand was under the eyelids; this lasted about two weeks, but rapidly disappeared under Prof. Roosa's treatment. About the same time her feet began to swell, the swelling gradually extending up to the knees. At this time she had a sharp pain in the precordial region which extended to the scapular region.

On account of great weakness, associated with nervousness, she was compelled to slide on the buttocks from step to step when coming down-stairs. There was a general feeling of pricking and a numbness which extended over the whole body. At times she would feel as if lost. Pains in the joints developed and were always increased prior to a storm.

When admitted to the hospital, the patient's general condition was poor; she was anæmic, fretful, childish, and unreasonable in her desires. Digestion good, bowels regular, but there was a constant desire to go to stool. Heart, lungs, temperature, and urine normal. Pupils persistently contracted to "pin-hole size;" they respond readily to distance, but not to light. There was a marked want of co-ordination in the muscular movements of the extremities, but without loss of power. There was a marked deformity of the left knee which consisted of a bulging on both sides of the joint, most noticeable upon the inner side. The prominence of the patella was absent. Manipulation showed that the superior extremity of the tibia could be dislocated in any direction. Accompanying the motions of the joint, there was heard a distinct crunching sound evidently produced by the rubbing together of the eroded bones which formed the articulation. An increase in the synovial fluid could not be detected. With all this swelling, deformity, and manipulation, there was a positive absence of pain. Before entering the hospital, the cavity of the joint was aspirated and four ounces of clear serous fluid withdrawn.

The right knee is somewhat deformed, but still maintains its contiguity.

Patient complained that she felt very nervous, and also of tingling throughout the body.

The pupils were dilated, and an opthalmoscopic examination made. No changes were discovered except a slight hyperæmia of the disk.

The treatment consisted in the internal administration of iodide of potassium, strychnia, and ergot, with cups to the back and faradization to the extremities.

Both plaster of Paris and the straight posterior splint was applied to the affected joints, with a slight improvement in the condition of the arthritic condition.

The patient remained in the hospital until February, 1885, the symptoms fluctuating, sometimes being worse, at others decidedly better. The right knee-joint grew worse, and the crunching sound became quite apparent when it was moved.

When readmitted to the hospital in February, 1886, all her symptoms were decidedly worse. Her general symptoms improved for a time, but the joint trouble still persisted, but without pain, although she suffered considerably from the general ataxic pains.

On the 20th of August, diarrhœa occurred, accompanied by increased pain, especially in the back. This, however, was easily controlled. But from this time on she grew steadily worse. On the 26th a decided induration was detected on the left buttock, apparently about the size of a walnut, the surface around this point being quite red. The pain was very severe, and morphine had to be administered.

On the morning of August 27th, the temperature rose to 105° F. without any chill, vomiting, or headache. The ataxia became very marked in every movement. The phlegmon on the left buttock had a very red and angry appearance, and the induration was found to be extending, but the œdema was apparently confined to the cellular tissue.

At one point the skin was black, and looked like a gangrenous slough. A small opening was found near the centre of the phlegmon, and a probe introduced which led into several sinuses. Cocaine was injected, and the sinuses laid open and packed with gauze. Temperature

at midnight, 103.4° F. Thirty grains of antipyrin was administered at 2 P.M. Temperature was down to 102.2° F. Restlessness subsided, and patient slept well.

August 28th. Pulse 126; temperature still high. Aug. 29th. Temperature still higher. Pus flowing freely from the wound. August 30th. Midnight temperature 102.8° F. Antipyrin, 30 grains. 4 A.M. Temperature 100.4° F. Pus was freely excreted by the wound. Pulse remained weak. Patient had some vomiting and a cough with pain in the region of the sternum. Breathing good, but subcrepitant, and mucous râles were heard. Sept. 1st. Temperature still elevated, and the pulse continued weak, although stimulants were freely administered. Sept. 2d. Redness, œdema, and increased heat was detected over a spot about two inches square on the right buttock. The redness faded gradually at the periphery of the œdema, and appeared to be due to changes in the connective tissue without involving the skin.

There was marked cerebral disturbance and great thirst.

The temperature was partially controlled by antipyrin.

Sept. 3d. Iodoform and cold cream applied to induration on right buttock. Mental condition nearly that of delirium. No repair apparent in the wound of the left buttock, but the surrounding induration was diminished.

Sept. 4th. No improvement. Pulse very rapid and weak. Urine red in color and acid in reaction. Sp. gr. 1.030, albumin, pus, and casts.

Sept. 5th. Patient was very restless, and soon became delirious, steadily sank and died comatose at 2.30 A.M., Sept. 6th, 1886.

The *necropsy* was made thirty-one hours after death. Rigor mortis was marked. Body abundantly supplied with adipose tissue. No external mark indicating a syphilitic lesion was found. The knees were diffusely enlarged by fluid within the joint cavity.

*Thoracic Cavity.*—The *pericardial* sac contained one-half ounce of blood-tinged serum. The *heart* was enlarged and its cavities dilated. The left ventricular wall was thickened and its muscular fibres degenerated. Weight, twelve

ounces. The segments of the bicuspid and aortic valves were nearly free from any fatty infiltration. The *left lung* was slightly adherent to the chest wall at the apex. The *right* was quite firmly bound to the chest-wall throughout by old pleuritic adhesions. Both lungs were congested and œdematous, with a tendency towards a hypostatic pneumonia. Weight of left, 10 ounces; of the right, 16 ounces.

*Abdominal cavity.*—The *spleen* was about normal in its general appearance. Weight, eight ounces.

*Kidneys.*—Both glands were enlarged. The left weighed six and one-half ounces; the right, six ounces. Their capsules were slightly thickened and adherent to the underlying renal tissue; the surface of the glands being slightly rough after enucleation. Their cortex was thickened, pale, and granular, and the markings wavy. Microscopic examination showed the chief lesion to be located in the epithelial cells, which were quite granular; there was also a slight interstitial thickening. The lesion was the hypertrophic form of chronic diffuse nephritis.

The *liver* was slightly enlarged and fatty. Weight, sixty-eight ounces.

*Cerebro-spinal System.*—The meninges of the brain and spinal cord were slightly thickened, and the pia mater of the brain was unusually pale and contained numerous thickened patches. The spinal cord was unusually soft at the time the necropsy was made, apparently from post-mortem change. But even in this softened condition, the wedge-shaped connective tissue development in the posterior columns was quite marked. Owing to the softened condition of the cord, it was difficult to properly harden the cord. The sections, however, which were obtained by Professor C. L. Dana, showed beyond doubt that the lesion was one of advanced tabes dorsalis.

The knee joints were opened and found to contain a large quantity of thick grumous pus which had distended the synovial sacs to such an extent as to cause rupture in both joints. The pus of the joint cavities had found its way into the cellular tissue and intermuscular planes and accounted

for the diffuse purulent infiltration of both thighs and the phlegmons found at the buttocks, the septicæmia, and death.

There were also extensive articular changes. There were from fifteen to twenty intra-articular cartilages in each joint cavity, some of which were attached to the synovial cavity by a small pedicle of fibrillated connective tissue, while others were loose and free in the cavities. Microscopic examination showed that they were composed of a dense fibrillated connective-tissue stroma, thickly interspersed with cartilage-cells. The articular surfaces of the bones entering into the formation of the knee joints also presented extensive change, the cartilage of incrustation being almost entirely destroyed, the underlying osseous structures having undergone extensive rarefying and formative osteitis with the development of small irregular, eburnated articular surfaces to replace the lost cartilaginous coverings. Between the projecting eburnated articular prominences, there were found numerous excavations evidently due to a carious process or a rarefying osteitis.

CASE II.—Mr. H. H., æt. 53, Austrian, widower, laborer, United States six years, was admitted to the Presbyterian Hospital August 26th, 1886.

*Previous History.*—The patient says that he was perfectly well and had never been sick until six weeks prior to his admission to the hospital, at which time he had peculiar sensations in his lower extremities, sometimes pricking, sometimes sharp shooting pains lasting only a short time, then disappearing to recur again and again. Has the same trouble in his arms and hands. Throws his feet up at the toes when he walks; ankles quite stiff. Cannot stand steady with his eyes closed, complained of a sensation in his feet as if walking on sand; this feeling is continuous.

*Physical examination.*—Apex beat of the heart in the fifth intercostal three inches from the median line; apex beat very feeble. A systolic murmur was heard at the apex and it was transmitted to the left. There was a diastolic murmur over the aortic valve, and it was transmitted downwards. Liver and spleen not enlarged. Trip-ham-

mer pulse, visible pulsation of the brachial and ulnar arteries. Absence of patellar reflex. Marked ataxic gait, accentuation of the heel movement in walking. Urine acid, specific gravity 1.025, no albumin and no casts.

Aug. 30th.—Patient had considerable pain, especially in the legs. Weight, 133 lbs.

No special change up to Sept. 10th. During the night he was apparently delirious, as he made several unsuccessful attempts to get up and dress. Sept. 11th his locomotion had become progressively worse, so that he can scarcely get about the ward without assistance.

During the day he became semi-stupid and lay in bed most of the time.

Sept. 12th.—During the past night his temperature, which had been previously normal, suddenly rose to 105.5° F., he began to cough and appeared to have considerable mucus in his bronchial tubes, which he could not raise. The pulse rose to 135 per minute and the respirations to 40, accompanied by a decided condition of stupor. Some crepitant râles were heard at the base of the right lung, and loud mucous râles all over both lungs. The vesicular murmur was roughened and high-pitched.

Stimulants were freely administered without effect.

Late in the evening he became semi-comatose, and muscular twitchings, especially of the arms, developed. They soon became violent enough to amount to general convulsions. Pupils equal; no signs of a cerebral hemorrhage or emboli.

Toward the morning of the 13th he became unable to swallow. He passed urine freely. The temperature remained high and he died comatose at 5 P.M. Sept. 18th, 1886.

*Necropsy*, forty-eight hours after death. Body fairly well supplied with adipose tissue. No decomposition changes, the body having been kept on ice. Muscles dark in color. Both legs between the knees and ankles were the seat of numerous white cicatrices the margins of which were raised and deeply pigmented as if of syphilitic origin.

*Thoracic cavity.*—The *pericardial sac* contained two

drachms of clear straw-colored serous fluid. There was one large milk patch upon the anterior surface of the right ventricle.

The *heart* was slightly enlarged, but its muscular substance was pale, soft, and flabby, and its cavities dilated. Springing from the acute margin there was a marked adipose fringe, and there was considerable adipose infiltration between the muscular planes. The mitral valve was but little changed by fatty infiltration. The segments of the aortic valve were extensively thickened and retracted by fatty infiltration. This condition, together with the dilatation of the left ventricular cavity, perfectly explains the aortic and mitral regurgitant murmurs heard during life. The muscular fibres showed fatty degeneration.

The *left lung* was quite firmly adherent to the chest-wall at the apex, and to the diaphragm at its base. The inferior lobe was the seat of numerous zones of lobular pneumonia in the stage of red hepatization, the remaining portion of the lung being congested and œdematous. Weight of left lung, twenty-eight ounces.

The *right lung* was quite adherent throughout by old and fibrous pleuritic adhesions. The inferior lobe showed still more marked evidence of lobular pneumonia, a large number of lobules being implicated, and some having reached the stage of gray hepatization. The remaining portions were congested and œdematous. Weight, thirty-three ounces.

*Abdominal cavity.*—The *spleen* was small, soft, and had a large fibrous patch upon its convex surface. Perisplenitis syphilitica. Weight of organ, five ounces.

*Kidneys.*—The left gland weighed six (6) ounces and the right five (5) ounces. Both kidneys showed distinctly the fœtal markings or lobulations. Their capsules were normal in thickness, and non-adherent to the underlying renal substance, the surface of which was perfectly smooth after enucleation. Their cortex was thickened and pale, but the markings were straight. The kidneys were practically normal. The *bladder* was over distended with urine.

The *liver* appeared to be normal. Weight, sixty ounces.

*Cerebro-spinal canal.*—The meninges of the brain and cord were thickened, especially the former. The dura mater was very firmly adherent to the pia mater and convexity of the brain along the margins of the longitudinal fissure. The pia mater was decidedly thickened and opaque throughout, numerous thickened patches also being found. No lesion of the cerebral substance was detected upon macroscopic examination.

When first removed, the spinal cord was firm and did not show any marked lesion, but after remaining a few days in the hardening fluid, it was plainly evident that there was a marked development of new fibrillated connective tissue in the posterior columns. Microscopic examination showed an advanced tabes dorsalis in the shape of atrophied nerve-fibres, and a decided increase in the fibrillated connective tissue of the posterior columns; this opinion being substantiated by the examination of the president, Professor Dana, who also examined the cord.

The two cases are interesting, as they illustrate a case of long duration with a complicating joint lesion, and one of unusually short duration.

The extensive suppurative arthritis without any pain directly referable to the joints involved is interesting.

The exact relation between the joint affection and the lesion of the cord is important. From the fact that joint affections are not, as a rule, associated with tabes dorsalis, it appears reasonable to look for the cause outside the lesion in the spinal cord, and to consider the joint affection more in the light of a coincident affection. As syphilis is a frequent cause for locomotor ataxia, and also for joint lesions, it seems reasonable to consider the syphilis as the etiological factor for both. The history, although not absolutely positive, points strongly in favor of a syphilitic infection a number of years back. The peculiar joint conditions are not those of an ordinary arthritis, but more strongly resemble the syphilitic lesions of the bones, in which there is a great tendency to have

both the formative and destructive process side by side. The changes are similar to the syphilitic osteoplastic periostitis with caries superficialis seen upon the surface of the long bones. It would appear, therefore, that the only connection between the two lesions was, that they both had the same cause.

It also shows that death was not caused by the cord lesion, but by the bursting of the capsules of the joints and the development of a diffuse suppurative cellulitis and septicæmia.

Exception may be taken to the short duration of the second case, but the history furnished by Dr. Alfred Edwards Hooker, house physician to the hospital, is undoubtedly correct.

It must be admitted by every one familiar with the pathological histology of posterior spinal sclerosis that a considerable development of new tissue and atrophy of the nerve fibres can be developed before symptoms are manifested.

In this particular case, the man always having been well and free from pains and aches, would be the one most likely to know had he suffered from them before his active symptoms developed. By a process of forced questioning, a series of symptoms might be developed which could be turned to account in making the history of longer duration. But in this instance even that resulted in a failure.

Although the patient's ataxia was rapidly progressing, his death was not directly attributable to it, but rather to the degenerated condition of his heart, and an acute broncho-pneumonia.

There appears to be no good reason why a patient could not develop some acute and complicating disease when the symptoms of locomotor ataxia are commencing and thus cause death early in tabes. Because a patient has symptoms of posterior spinal sclerosis for a few weeks, and then develops a pneumonia and dies is a poor reason for insisting that he must have had symptoms for a year or two without knowing it.

In fact, it is pretty generally admitted that locomotor ataxia in itself rarely, if ever, causes death ; but death, when it does occur, is always the result of some secondary and complicating condition. Why might not the complication be developed in the first week of the ataxia, and in this way render the disease of short duration?